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Articles

Effect of Continuous Positive Airway Pressure on Intrathoracic and Left Ventricular Transmural Pressures in Patients With Congestive Heart Failure

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Abstract

Background

Continuous positive airway pressure (CPAP) can improve cardiac function in patients with congestive heart failure (CHF). We hypothesized that this effect might be related to CPAP-induced increases in intrathoracic pressure, which would reduce left ventricular transmural pressure (LVP_{tm}) during systole, thereby decreasing left ventricular afterload.

Methods and Results The effect of graduated CPAP from 0 to 10 cm H_2O on the above variables was examined over a 75-minute period and compared with a 75-minute time control period without CPAP in two groups of subjects: 15 patients with CHF and 9 healthy subjects. Intrathoracic pressure was estimated from esophageal pressure (P_{es}), and systolic LVP_{tm}, a determinant of left ventricular afterload, was assessed by subtracting P_{es} during systole from systolic blood pressure. Cardiac index (CI) was assessed by Doppler echocardiography. At baseline, inspiratory P_{es} amplitude, which reflects inspiratory muscle force generation, was greater in the patients with CHF than in the healthy group (9.9±0.8 versus 5.5±0.4 mm Hg, $P<.001$). In addition, systolic P_{es}, which represents the relative contribution of intrathoracic pressure to LVP_{tm}, was more negative in the patients with CHF than in the healthy group (-4.1±0.3 versus -2.2±0.1 mm Hg, $P<.001$). While on CPAP of 10 cm H_2O, inspiratory P_{es} amplitude decreased and systolic P_{es} increased significantly in the group with CHF (from 11.1±1.1 to 7.5±1.1 mm Hg, $P<.025$ and from -4.7±0.6 to 0.6±0.6 mm Hg, $P<.001$, respectively), but CPAP had no effect on these variables in the healthy subjects. Compared with the equivalent time control period, P_{es} amplitudexrespiratory rate decreased significantly while on CPAP in both the group with CHF (from 188±22 to 112±17 mm Hgxbreaths per minute, $P<.005$) and the healthy group (from 82±8 to 60±6 mm Hgxbreaths per minute, $P<.05$). Compared with time control, systolic LVP_{tm} decreased significantly while on CPAP, from 116.0±5.3 to 110.3±4.5 mm Hg ($P<.025$) in the group with CHF, but did not change in the healthy group. Moreover, systolic LVP_{tm}xheart rate decreased significantly in the group with CHF (from 80.55±5.27 to 71.83±4.73 mm Hgxbeats per minute/100, $P<.005$) but not in the healthy group. CI decreased significantly while on CPAP in the healthy group ($P<.025$) but did not change in the group with CHF.

Conclusions In patients with CHF, the inspiratory muscles generate greater force per breath and systolic P_{es} contributes more to LVP_{tm} than in healthy subjects. By increasing intrathoracic pressure in patients with CHF, CPAP unloaded inspiratory muscles and reduced left ventricular afterload without compromising CI.

Key Words: ventricles • pressure • heart failure

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Continuous positive airway pressure (CPAP) administered via a face or nasal mask can acutely augment cardiac output in patients with poorly compensated congestive heart failure (CHF).[1] [2] Chronic improvements in left ventricular ejection fraction (LVEF) have also been reported with nightly application of CPAP in patients with CHF and coexistent obstructive or central sleep apnea.[3] [4] [5] It has been postulated that CPAP exerts its effects on cardiac performance by increasing intrathoracic pressure and thereby reducing cardiac preload, by impeding cardiac filling, and afterload, by reducing left ventricular transmural pressure (LVP_{tm}).[1] [2] [6] [7] Reductions in preload and afterload in response to CPAP have been documented in experimental animals with healthy cardiac function as demonstrated by decreases in left ventricular end-diastolic and end-systolic volumes.[7] [8] [9] There also is evidence that CPAP can reduce heart rate in patients with CHF.[2] [10] However, the effect of CPAP on intrathoracic pressure and left ventricular afterload in humans with CHF has not been determined.

Vasodilators exert their beneficial effects on cardiac function in CHF primarily by reducing left ventricular afterload secondary to reducing vascular resistance.[11] [12] If CPAP also reduces left ventricular afterload in patients with chronic CHF, this effect might contribute to improved cardiac function.[1] [2] [3] [4] We hypothesized that because CPAP raises intrathoracic pressure, it should reduce LVP_{tm} during systole (the difference between left ventricular systolic pressure and extracardiac pressure)[6] and thereby reduce left ventricular systolic wall stress. If CPAP simultaneously reduces heart rate in patients with CHF, it should reduce pressurexheart rate, an important determinant of myocardial O_2 consumption.[10] [13] Therefore, the objectives of the present study were to determine the effects of CPAP on intrathoracic pressure, LVP_{tm}, and LVP_{tm}xheart rate in patients with CHF and in healthy subjects.

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Subjects

Volunteers free of cardiac or respiratory disease were recruited as healthy subjects. Patients less than 75 years of age with chronic CHF were recruited on the basis of a history of CHF secondary to ischemic or idiopathic dilated cardiomyopathy for at least 6 months, chronic exertional dyspnea despite medical therapy, and a left ventricular ejection fraction <45% measured by ^{99}Tc equilibrium radionuclide angiography. The protocol was approved by the Ethics Committee for Human Experimentation at the University of Toronto, and all subjects gave consent before entering the

study.

Arterial and Esophageal Pressures

Blood pressure was measured every minute with an automatic sphygmomanometer (PhysioControl Lifestat 200). Heart rate was derived from a precordial ECG lead. Esophageal pressure (P_{es}) was measured using a balloon catheter system attached to a pressure transducer (45±50 cm H_2O, Validyne MP) placed in the esophagus according to the method of Baydur et al[14] such that change in P_{es} equaled change in mouth pressure during occluded breaths. Respiratory movements were detected by a pneumobelt from which respiratory rate was calculated. ECG, P_{es}, and pneumobelt output were recorded continuously onto a strip-chart recorder (model 2800S, Gould).

Stroke Volume and Cardiac Output

Stroke volume and cardiac output were determined by Doppler echocardiography (Ultramark 8, Advanced Technology Laboratories) as previously described for our laboratory.[15] With patients in the supine position, maximum instantaneous aortic flow velocity was measured in the ascending aorta using continuous wave Doppler (2.25 MHz) directed through the suprasternal window. Stroke volume was calculated from the product of the mean time-velocity integral and the cross-sectional area of the aortic annulus orifice (A): $A=\pi(D/2)^2$, where D is the diameter of the aortic annulus obtained from a prior parasternal long-axis view at baseline. In addition, in four healthy subjects and four patients with CHF, aortic annular diameter was determined during the application of 10 cm H_2O of CPAP. Aortic annular diameter did not change from baseline to CPAP of 10 cm H_2O in either the healthy subjects (from 2.04±0.06 to 2.04±0.05 cm) or the patients with CHF (from 1.71±0.08 to 1.72±0.08 cm). We therefore assumed no change in aortic annular area in those receiving CPAP. Integrals for 30 to 60 heartbeats were averaged to obtain a mean time-velocity integral that, by taking into account inspiratory and expiratory phases, would represent an average over the entire respiratory cycle. Cardiac output was calculated from the product of stroke volume and heart rate. Stroke volume index (SVI) and cardiac index (CI) were then derived taking into account the body surface area.

Protocol

Healthy subjects and patients with CHF were studied during a time control period and while receiving CPAP (BiPAP, Respironics Inc) for a 75-minute period, while awake and supine on two consecutive afternoons in random order. Thus, both healthy subjects and patients with CHF acted as their own controls. On the time control day, after a 15-minute baseline recording period without a mask on, an open mask with a 3-cm lumen of negligible resistance was placed over each subject's nose with the mouth closed for 45 minutes and then was removed for 15 minutes. On the CPAP day, after a 15-minute baseline period, CPAP was applied via a tightly fitting nasal mask with the mouth closed for 15 minutes at each of three consecutive pressures—5, 7.5, and 10 cm

H_2O (3.7, 5.5, and 7.4 mm Hg, respectively)—followed by a 15-minute recovery period without CPAP.

Statistical Analysis

End-expiratory P_{es} during the initial recording period on each of the 2 days was considered the baseline value.[16] The average end-expiratory P_{es}, amplitude of end-expiratory P_{es} to peak inspiratory P_{es}, peak inspiratory P_{es} compared with baseline end-expiratory P_{es}, and P_{es} during cardiac systole measured synchronously with the QRS complex of the ECG were calculated from the last 2 minutes of each 15-minute interval. Although maximum left ventricular systolic pressure is generated between the QRS and T waves, the QRS is a more distinct reference point for systole and provides a convenient method of determining an appropriate sampling frequency for the determination of systolic LVP_{tm} and systolic LVP_{tm}xheart rate. This sampling method also takes into account the exposure of the left ventricle to P_{es} during both inspiration and expiration to give an average systolic P_{es} throughout the respiratory cycle (Fig 1). Since brachial artery systolic pressure accurately reflects left ventricular end-systolic pressure in the absence of aortic outflow obstruction, LVP_{tm} values at the peak of inspiration and during cardiac systole were calculated by subtracting peak inspiratory P_{es} and systolic P_{es} (ie, the most negative and mean P_{es} values to which the heart is exposed during systole), respectively, from the average brachial systolic blood pressure during each time interval. Systolic LVP_{tm}xheart rate was used as another index of afterload that adjusts for changes in heart rate.[10] [13] The P_{es} amplitudexrespiratory rate product was used as an index of inspiratory muscle force generation and energy requirement over time.[17] [18]

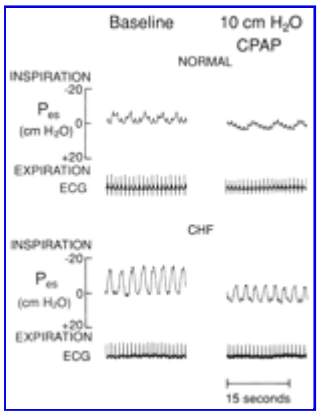

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Figure 1. Esophageal pressure (P_{es}) and ECG recordings from a healthy subject (normal) and a patient with congestive heart failure (CHF) at baseline (left) and on 10 cm H_2O of continuous positive airway pressure (CPAP) (right). Note that in the healthy subject, CPAP caused both end-expiratory P_{es} and peak inspiratory P_{es} to become more positive but caused no change in the amplitude of the inspiratory P_{es} swing. Respiratory rate also decreased. In the patient with CHF, the P_{es} amplitude at baseline is greater than in the healthy subject. On CPAP, end-expiratory P_{es} becomes more positive and P_{es} amplitude is reduced, such that peak inspiratory P_{es} becomes more positive. Respiratory rate did not change.

Comparisons between data for the healthy group and for the group with CHF at baseline were made using two-tailed unpaired *t* tests. Two-way ANOVA for repeated measures was used to compare variables on the CPAP day with the equivalent time control period within both the healthy and CHF groups. For the purposes of clarity, *P* values reported in the text refer to comparisons at 10 cm H_2O of CPAP and the equivalent time control period unless otherwise stated. A value of $P<.05$ was considered statistically significant. Results are expressed as mean±SEM.

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Subject Characteristics

Nine healthy subjects and 15 patients with CHF were studied. Ten patients had ischemic and 5 had idiopathic dilated cardiomyopathy. One CHF patient was female but in all respects responded to CPAP in a fashion similar to the male patients. Twelve patients were in sinus rhythm and 3 were in atrial fibrillation. Left ventricular ejection fraction in the group with CHF was reduced at 17.9±2.4%. The patients with CHF were normoxic and eucapnic (PaO_2 of 82.3±3.1 mm Hg and $PaCO_2$ of 39.8±2.2 mm Hg). Vasodilators were being taken by all patients, digoxin by 66%, and diuretics by 93%.

The characteristics of the subjects are shown in the Table. The groups were comparable for body mass index and baseline respiratory rates. P_{es} amplitude, peak inspiratory P_{es} (which, at baseline, is identical to P_{es} amplitude), and P_{es} amplitudexrespiratory rate were significantly greater ($P<.001$), whereas systolic P_{es} was significantly more negative in the group with CHF than in the healthy group ($P<.001$). The healthy group and the group with CHF mean heart rates and diastolic blood pressures were comparable. However, as one would expect, the group with CHF had a lower systolic blood pressure, SVI, and CI ($P<.001$) compared with the healthy group. Although the healthy subjects were significantly younger than the patients with CHF ($P<.001$), a comparison of P_{es} amplitude, which is the variable showing the greatest difference between the two groups and is the variable of greatest interest, in the four youngest and four oldest healthy subjects (age, 31.8±0.9 versus 53.3±4.3 years) revealed no significant difference (5.7±0.8 versus 6.5±0.9 mm Hg, $P=.539$). Similarly, within the group with CHF, the P_{es} amplitude of the four youngest and four oldest patients (53.5±1.3 versus 65.3±1.9 years) was not significantly different (9.9±2.3 versus 8.5±1.1 mm Hg, $P=.968$). In addition, there was no significant correlation between age and P_{es} amplitude in either the healthy group ($r=.203$, $P=.60$) or the group with CHF ($r=-.255$, $P=.40$) or within the combined healthy and CHF groups ($r=.265$, $P=.23$). Accordingly, there was no significant effect of age on P_{es} amplitude, and therefore differences between the healthy subjects and the patients with CHF for this variable are attributable to disease state. The

peak inspiratory LVP_{tm}, systolic LVP_{tm}, and systolic LVP_{tm}xheart rate at baseline were comparable between groups.

View this table: **Table 1.** Baseline Characteristics of Study Subjects
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Effects of CPAP

Baseline values for all variables were comparable on the time control and CPAP days within the healthy and CHF groups. During the time control, all variables remained constant over time in both groups. In the post-CPAP recovery period, all variables returned to the time control level except heart rate, which remained lower in the group with CHF.

Healthy subjects experienced an increase in end-expiratory P_{es} of 3.0 mm Hg ($P<0.001$) but no change in the P_{es} amplitude (Figs 1 and 2). Peak inspiratory and systolic P_{es} both increased by 4.1 mm Hg ($P<.005$) (Fig 3). Patients with CHF experienced a 2.9 mm Hg increase in end-expiratory P_{es} ($P<.001$), a 3.6 mm Hg reduction in mean P_{es} amplitude ($P<.025$), a 6.1 mm Hg increase in peak inspiratory P_{es} ($P<.001$), and a 5.3 mm Hg increase in systolic P_{es} ($P<.001$) (Figs 1 through 3). Respiratory rate decreased in the healthy group (-1.6 breaths per minute, $P<.01$) but not in the group with CHF (Fig 4). P_{es} amplitudexrespiratory rate decreased by 27% in those receiving CPAP in the healthy group (-22 mm Hgxbreaths per minute, $P<.05$) and by 40% in the group with CHF (-76 mm Hgxbreaths per minute, $P<.005$).

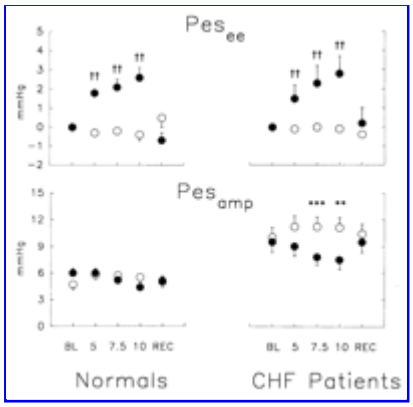

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Figure 2. Plots of group data for end-expiratory esophageal pressure (Pes_{ee}) and esophageal pressure amplitude (Pes_{amp}) in healthy subjects (normals) and patients with congestive heart failure (CHF) at 15-minute time intervals: baseline (BL), 5 cm H_2O (5), 7.5 cm H_2O (7.5), 10 cm H_2O (10), and recovery (REC) during time control (open circles) and continuous positive airway pressure (CPAP) (closed circles) days. This figure illustrates that during the time-control day, variables remained stable, and that on the CPAP day, variables returned to the baseline values during the recovery period. Note especially the CPAP dose-related increase in Pes_{ee} and reduction in Pes_{amp} in the patients with CHF. $^{**}P<.025$, $^{***}P<.01$, ‡$P<.001$.

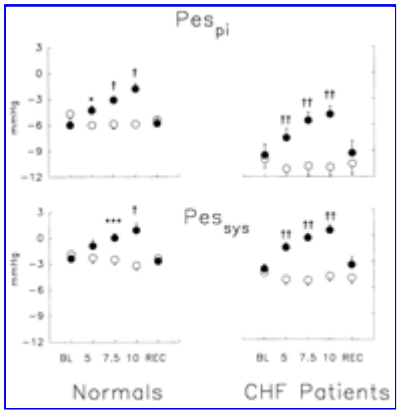

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Figure 3. Plots of group data for peak inspiratory esophageal pressure (Pes_{pi}) and systolic esophageal pressure (Pes_{sys}). With progressively higher CPAP level, both variables become progressively more positive. See Fig 3 for abbreviations. *P<.05, †P<.005; otherwise as for Fig 3.

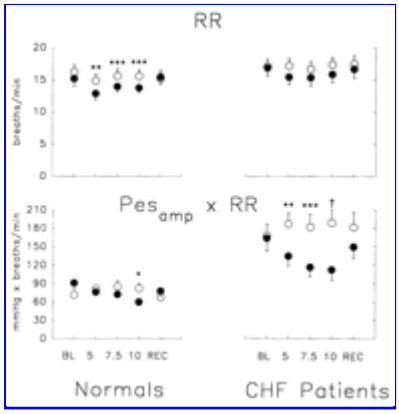

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Figure 4. Plots of group data of the respiratory rate (RR) and the product of Pes_{amp} and RR. RR decreased at all levels of CPAP in the healthy subjects but not in the patients with CHF. There was a CPAP dose-related decrease in Pes_{amp}xRR that was more pronounced in the patients with CHF than in the healthy subjects. See Fig 3 for abbreviations.

Systolic and diastolic blood pressures did not change significantly on CPAP in either group (Fig 5). Because increases in peak inspiratory and systolic P_{es} were offset by a slight increase in systolic blood pressure in the healthy subjects, peak inspiratory and systolic LVP_{tm} (Fig 6) on CPAP did not decrease. In contrast, the group with CHF experienced reductions in both peak inspiratory and systolic LVP_{tm} (-6.1 mm Hg, P<.01, and -5.7 mm Hg, P<.025, respectively). Heart rate decreased with CPAP only in the group with CHF (-5.2 beats per minute, P<.01, Fig 7). Consequently, systolic LVP_{tm}xheart rate decreased significantly by 11% (-8.7 mm Hgxbeats per minute/100, P<.005) in the CHF group but not in the healthy group.

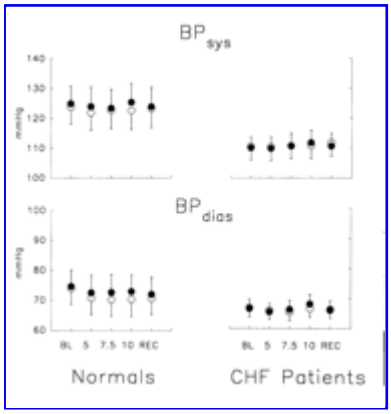

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Figure 5. Plots of group data for systolic (BP_{sys}) and diastolic (BP_{dias}) blood pressures. There was no change in either of these variables over time or while receiving CPAP in either group. See Fig 3 for abbreviations.

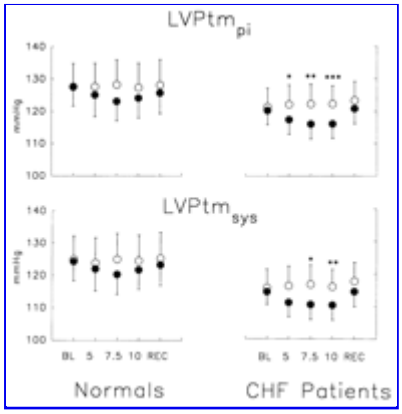

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Figure 6. Plots of left ventricular transmural pressure gradient during peak inspiration ($LVPtm_{pi}$) and cardiac systole ($LVPtm_{sys}$). There are CPAP dose-related reductions in both these variables in the group with CHF but not in the healthy group. See Fig 3 for abbreviations.

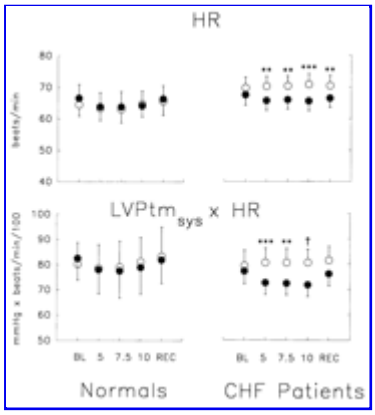

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Figure 7. Plots of heart rate (HR) and the product of $LVPtm_{sys}$ and HR ($LVPtm_{sys}$xHR). CPAP dose-related reductions in both HR and $LVPtm_{sys}$ are seen in the patients with CHF but not in the healthy subjects. See Fig 3 for abbreviations.

SVI and CI decreased while on CPAP from 34.2±1.7 to 30.5±1.0 mL/m^2 (P<.01) and from 2.20±0.14 to 1.96±0.14 L · min^{-1} · m^{-2} (P<.025), respectively, among healthy subjects (Fig 8). In contrast, SVI and CI did not change in the patients with CHF while on CPAP. However, there was considerable variation in the response, with approximately half experiencing an increase and half experiencing a decrease in CI and SVI, as seen in previous studies.[1] [2]

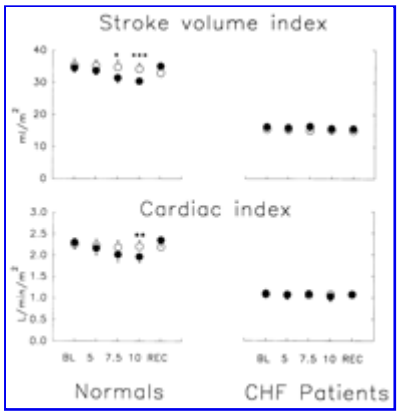

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Figure 8. Plots of stroke volume and cardiac indexes. There are progressive decreases in both stroke volume and cardiac indexes on progressively higher levels of CPAP in the healthy group but not in the group with CHF. See Fig 3 for abbreviations.

Discussion

The primary purpose of the present study was to assess the effects of CPAP on intrathoracic pressure, as reflected by P_{es}, and on left ventricular afterload, as reflected by LVP_{tm}, in healthy subjects and in patients with CHF. Three main

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observations were made. First, the amplitude of inspiratory P_{es} swings was greater in patients with CHF than in healthy subjects. These data indicate that inspiratory muscles of patients with CHF generate greater force than those of healthy subjects.[17] [19] [20] Second, the more negative systolic P_{es} in the group with CHF contributed proportionally more to systolic LVP_{tm} than it did in the healthy subjects. Third, among patients with CHF but not healthy subjects, increasing levels of CPAP were associated with progressive decreases in P_{es} amplitude, peak inspiratory and systolic LVP_{tm}, and heart rate. CI did not change in patients with CHF. The clinical implications of our findings are that CPAP unloads the left ventricle and inspiratory muscles of patients with CHF without compromising CI.

Effects of CPAP on P_{es}

LVP_{tm} is an important determinant of left ventricular afterload. In the present study, we used P_{es} to estimate extracardiac pressure[21] [22] [23] and to calculate peak inspiratory and systolic LVP_{tm}.[6] [24] Changes in P_{es} have been shown to correlate closely with changes in pleural and pericardial pressures during systole under a variety of conditions.[21] [22] Therefore, changes in P_{es} provide a reasonable estimate of change in intrathoracic[14] [18] and systolic pericardial pressures, if not their absolute magnitudes. P_{es} amplitude provides a good index of the degree of inspiratory muscle force generation and energy expenditure per breath.[17] [18] The greater P_{es} amplitude observed in patients with CHF agrees with previous findings[19] and probably contributes to dyspnea.[17] [19] [20] In the present study, we used P_{es} amplitudexrespiratory rate as an index of inspiratory force generation over time.[17] [18] We found that P_{es} amplitudexrespiratory rate was significantly greater in the group with CHF than in the control group. These findings indicate that the inspiratory muscles of patients with CHF generate more force and would require a greater proportion of a lower CI to meet their increased energy demands than would those of healthy subjects.[25]

Two likely explanations for the high P_{es} amplitude in the group with CHF were that lung compliance was reduced or airway resistance increased due to interstitial and bronchial edema.[8] [19] [26] In addition, the large P_{es} amplitude may have reflected a vagally mediated increase in ventilatory drive secondary to elevated pulmonary venous pressure.[27] However, because we did not measure tidal volume or compliance, we cannot discuss the relative contribution of each of these factors to increased P_{es} amplitude.

The greater reduction in P_{es} amplitude in those receiving CPAP in the group with CHF than in the healthy group is interesting and suggests that the impact of CPAP may be amplified in the presence of increased lung water. This could occur, for example, if CPAP caused an extrathoracic redistribution of lung water leading to an increase in lung compliance[7] [28] [29] [30] or if it caused a

reduction in tidal volume or an increase in lung volume to a more favorable position on the pressure-volume curve.[28] [31] Regardless of the mechanism, CPAP-induced reductions in P_{es} amplitude and P_{es} amplitudexrespiratory rate indicate that the inspiratory muscles were unloaded, thus reducing their energy demands.[18] [28] [32]

Effects of CPAP on LVP_{tm}

Negative intrathoracic pressure contributes to left ventricular afterload by increasing LVP_{tm}.[6] [33] [34] Therefore, the more negative systolic P_{es} in the group with CHF had a relatively greater impact on systolic LVP_{tm} than in the healthy group. In contrast to the healthy heart, the failing heart is very sensitive to changes in afterload, so hemodynamics are more adversely affected by exaggerated negative intrathoracic pressure.[23] [33] [34] [35] [36] For example, exaggerated negative intrathoracic pressure during obstructive sleep apneas probably contributes to impaired myocardial function in patients with dilated cardiomyopathy.[3] Accordingly, the most important finding of the present study was that under carefully controlled conditions, CPAP caused consistent dose-related reductions in peak inspiratory and systolic LVP_{tm} in the group with CHF secondary to increases in peak inspiratory and systolic P_{es}. This contrasted with the lack of an effect on LVP_{tm} in healthy subjects, where small increases in P_{es} while on CPAP were offset by slight increases in systolic blood pressure (Figs 3, 5, and 6). What is remarkable is that these effects of CPAP in the group with CHF were observed in addition to afterload reduction by pharmacological agents.[37] [38] Moreover, reductions in LVP_{tm} secondary to CPAP resulted from increases in P_{es} rather than decreases in systolic blood pressure. This may well be one of the mechanisms responsible for the long-term improvements in cardiac function we observed after nightly application of CPAP to medically treated patients with CHF and coexisting sleep apnea.[3] [4] [5]

The group with CHF experienced a significant fall in heart rate while receiving CPAP.[2] [10] This suggests that CPAP either reduced cardiac sympathetic or increased parasympathetic activity, directly or reflexively. However, a decrease in intrathoracic aortic transmural pressure due to CPAP would tend to decrease baroreceptor discharge. In addition, neither CI nor blood pressure changed in the group with CHF while receiving CPAP. Consequently, it is more likely that stimulation of pulmonary vagal afferents by CPAP-induced lung inflation increased parasympathetic tone and reflexively reduced cardiac sympathetic outflow.[39] In contrast, in the healthy group, the sympathostimulatory effects of a reduced CI while receiving CPAP probably offset any sympathoinhibitory effects of lung inflation and accounted for the absence of any net change in heart rate.

CPAP caused reductions in SVI and CI in the healthy group but did not reduce afterload. Therefore, decreases in CI probably arose from CPAP-induced reductions in preload.[7] [9] [22] [40] [41] In

contrast, the group with CHF did not experience a decrease in SVI or CI while receiving CPAP. This finding is consistent with previous work showing that positive pressure breathing increases SVI and CI in patients with CHF and elevates left ventricular filling pressures but reduces SVI and CI in those whose filling pressures are normal.[2] [42] [43] [44] Approximately half of our patients with CHF experienced an increase in SVI and CI and the remainder experienced a decrease, resulting in no net change in SVI or CI. Because cardiac filling pressures were not measured, patients could not be separated into those with high and low preloads. However, a reasonable explanation for the absence of any increase in group mean values for SVI and CI in the patients with CHF is that the preload-reducing effect of CPAP offset the afterload-reducing effect in patients with low filling pressures.

Doppler estimates of stroke volume have been validated under experimental conditions similar to ours.[41] [45] Estimations of stroke volume by continuous wave Doppler yield measures of stroke volume that are as reliable and reproducible as those obtained by pulsed wave Doppler.[46] In addition, we have shown that 10 cm H_2O of CPAP does not affect aortic annular diameter, so time-velocity integrals measured in those receiving CPAP are unlikely to have been subject to artifactual influences. Although Doppler measurements tend to systematically underestimate the absolute stroke volume,[47] they accurately reflect changes in stroke volume.[41] [45] [46] [47] [48] [49] Therefore, changes in SVI in response to CPAP in our subjects likely provide a reasonable estimate of the magnitude of change in this variable. Among the patients with CHF, CPAP caused a reduction in systolic LVP_{tm}xheart rate (an index of myocardial systolic force generation and O_2 consumption over time[10] [13]). This effect, combined with the reduced P_{es} amplitudexrespiratory rate in those receiving CPAP, will reduce myocardial and inspiratory muscle energy demands, allowing redistribution of blood flow to other organs to better match their energy requirements. Moreover, the reduction in heart rate in the group with CHF could potentially improve subendocardial perfusion and allow for better left ventricular diastolic filling. Therefore, CPAP can improve cardiorespiratory efficiency in patients with CHF even in the absence of any increase in CI.

In summary, we have demonstrated that by increasing intrathoracic pressure, CPAP reduces left ventricle afterload and unloads the inspiratory muscles of patients with CHF. These findings may help to explain short- and long-term improvements in cardiorespiratory function that have been reported in patients with CHF while receiving CPAP.[3] [4] [5] [10] Further physiological studies will be required to determine other mechanisms whereby CPAP exerts these effects. Our findings also provide a strong rationale for examining the clinical usefulness of CPAP as a nonpharmacological method of reducing afterload in patients with CHF.

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